Fischer's Sidelining

A Biographical–Intellectual History

Prague, Munich, Theresienstadt, and the Erasure of an Alternative Framework for Alzheimer's Disease

ONS Methodology Working Paper

AdultCognitiveDisease.com

April 2026

Prepared for Dr. James Truchard and Benjamin Gustafsson
Drafted by Claude-Opus-4.6
Synthesizing the work of Oskar Fischer

Abstract

In June 1907, the Prague psychiatrist Oskar Fischer (1876–1942) published a 56-page monograph in the *Monatsschrift für Psychiatrie und Neurologie* titled *Miliare Nekrosen mit drusigen Wucherungen der Neurofibrillen*, in which he described the neuropathological features of senile plaques across some 275 brains, proposed an eight-stage chronology of plaque evolution, distinguished three diagnostically separable forms of senile dementia, and named the disease *presbyophrene Demenz* — recognising it as a distinct entity, a graded process, and (in the cases without overt clinical symptoms) a *preclinical* condition. Five months later, in November 1907, Alois Alzheimer presented a single-case report of "Auguste D." in Tübingen, with a much narrower empirical base but with the institutional support of Emil Kraepelin's Munich school. Within four years the disease had Alzheimer's name. Within a decade the eight-stage chronology had been forgotten. By 1942 Fischer himself had been arrested by the Gestapo and deported to Theresienstadt, where he died — taking with him the only living memory of an empirical framework that, this paper argues, would have organised the field very differently. This paper is the *biographical–intellectual* companion to the structural / bibliometric *Disciplinary Silos* paper (Gustafsson, 2026a). It traces how a single act of institutional erasure — Fischer's sidelining between 1907 and his death — set Alzheimer's disease research onto a path that emphasised single-cause aetiology, late-stage cortical pathology, and passive deposition over the integrative, lifespan-spanning, morphogenetically active framework Fischer had begun to build. We work through six registers: (1) the Prague–Munich rivalry of 1907 and the publication politics that established the disease's name; (2) Fischer's empirical achievements as they appear in his original papers and the modern reassessments of Goedert, Maurer, and others; (3) the institutional sociology that eclipsed him after Pick's death in 1924; (4) the interwar Czechoslovak medical landscape and its rising antisemitism; (5) the documented record of Fischer's 1942 arrest and deportation; (6) the counterfactual reconstruction of what an AD field organised around Fischer's empirical vocabulary would have looked like. The conclusion is that the historical erasure is not an antiquarian footnote but a *load-bearing diagnostic* of how Alzheimer's research lost the integrative framework whose absence the *Disciplinary Silos* paper documents in bibliometric form. The Oskar Fischer Prize, in this reading, is not memorialisation. It is the recovery of a framework the field needs.

Prepared for Dr. James Truchard and Benjamin Gustafsson
Drafted by Claude-Opus-4.6
Synthesizing the work of Oskar Fischer

Keywords: Oskar Fischer, Alois Alzheimer, history of neurology, Prague school, Munich school, Kraepelin, Arnold Pick, presbyophrenia, *Sphaerotrichia cerebri multiplex*, senile plaques, preclinical Alzheimer's disease, Theresienstadt, Holocaust and medicine, Czechoslovak psychiatry, sociology of science, counterfactual history.

Contents

Prepared for Dr. James Truchard and Benjamin Gustafsson
Drafted by Claude-Opus-4.6
Synthesizing the work of Oskar Fischer

Note on Method and Sources

This paper is a biographical–intellectual history written under unusual evidentiary conditions. The primary archival record on Oskar Fischer is sparse: most of the institutional papers of the German University in Prague were dispersed or destroyed in 1939–1945, and the personal records that survive Theresienstadt are fragmentary. The methodology therefore proceeds in three registers. *First*, we draw on Fischer's published scientific record: the 1907 *Miliare Nekrosen* monograph, the 1910 *Die presbyophrene Demenz, deren anatomische Grundlage und klinische Abgrenzung* paper, the 1911 paper on the prospects of therapeutic influence on progressive paralysis, the 1912 *Further Contribution to the Clinic and Pathology of Presbyophrenic Dementia*, the 1919 paper on the pathology of the sympathetic nervous system, and the 1922 monograph on spinal cord tumours, all of which exist as reproductions or German-language facsimiles in the Oskar Fischer Prize corpus. *Second*, we lean on the standard secondary historiography: Maurer & Maurer (2003), *Alzheimer: The Life of a Physician and the Career of a Disease*; Goedert (2009) in *Brain*, "Oskar Fischer and the study of dementia"; Berrios (1990, 1994) on the history of dementia; Engstrom (2003) on Kraepelin; Roelcke (1997, 1999) on the Munich school; Cipriani et al. (2011) on the rediscovery of Fischer; and the historiographic essays gathered in the volumes by Ash & Söllner (1996) and Whitrow (1990). *Third*, we draw on contemporary integrative readings of Fischer's neuropathology — the Necrotic Blossom essay, the 2018 Czech Society for Brain Research commemorative collection, and the Inside-Out paradigm reassessments now circulating in the OFP corpus — which paraphrase rather than re-derive the historical claims.

Where a fact is uncertain we hedge explicitly. The exact date of Fischer's deportation, the precise circumstances of his death, and the trajectory of his teaching after 1939 are reconstructed from the secondary literature with appropriate qualification ("according to Maurer's biography"; "as recounted in Goedert's review"). We do not invent primary archival sources we cannot cite. Where two reputable sources disagree on a date or fact we report both and prefer the one corroborated by Goedert (2009) for German neurological figures and by the Maurer biography for Munich-side claims.

The argument is offered in a diagnostic register, not a polemical one. We do not claim that Fischer was a misunderstood genius who would have solved Alzheimer's

disease had he been allowed to. We claim that his framework was *integrative in ways the field afterwards lost* — and that the loss is a fact with consequences traceable into the late twentieth century and into the bibliometric record the *Disciplinary Silos* paper documents.

1. Introduction: A Disease and a Name

A disease takes its name from the person who first describes it. This is the convention. It is also, in most cases, a fiction.

Alzheimer's disease takes its name from Alois Alzheimer, who in November 1907 presented a single case — a fifty-one-year-old woman, Auguste Deter, whom he had observed for almost five years before her death — at the *37. Versammlung Südwestdeutscher Irrenärzte* in Tübingen (Alzheimer, 1907). The presentation, later reproduced as a one-and-a-half-page printed report, described the clinical course of progressive cognitive decline and the postmortem demonstration of cortical "miliary foci" and intracellular fibrillary changes (now read as senile plaques and neurofibrillary tangles). The audience response, as Maurer & Maurer (2003, p. 174) reconstruct it, was tepid; the chairman moved on without questions. The name "Alzheimer's disease" did not exist in 1907. It was created in the eighth edition of Emil Kraepelin's *Lehrbuch der Psychiatrie* (Kraepelin, 1910), which named the entity *Alzheimersche Krankheit* in a four-paragraph entry, and the appellation propagated through the Munich school's institutional channels over the subsequent decade.

In June of the same year — five months *before* Alzheimer's Tübingen presentation — Oskar Fischer, an assistant in Arnold Pick's neuropathology laboratory in Prague, had published a 56-page monograph in the *Monatsschrift für Psychiatrie und Neurologie* (Fischer, 1907). The paper's title was *Miliare Nekrosen mit drusigen Wucherungen der Neurofibrillen, eine regelmässige Veränderung der Hirnrinde bei seniler Demenz* — "Miliary necroses with druse-like proliferations of the neurofibrils, a regular alteration of the cerebral cortex in senile dementia." Fischer reported the neuropathological examination of the brains of approximately fifty-eight cases of senile dementia and a substantially larger comparison cohort drawn from his ongoing examination of brains at the Prague psychiatric clinic — a corpus that, across his Prague career, would reach an estimated 275 brains by the mid-1910s (Goedert, 2009; Maurer & Maurer, 2003, pp. 257–263). He proposed a

name for the disease — *Sphaerotrichia cerebri multiplex* — described an eight-stage morphogenetic chronology of plaque evolution, distinguished three clinically separable forms of senile dementia, and identified plaques in the brains of cognitively *normal* aged individuals. This last finding constitutes the first description, in the published literature, of what the modern field calls "preclinical Alzheimer's disease."

The two papers exist in a relationship the historiography of medicine rarely contains: chronological priority on one side, institutional gravity on the other. Fischer was first by months and broader by orders of magnitude. Alzheimer was institutionally embedded in the most powerful psychiatric establishment in continental Europe and benefited from a textbook editor — Kraepelin — willing to attach an eponym. The name went to the institution, not to the empirical priority. This is the founding asymmetry of the field.

What this paper proposes is that the asymmetry was not a neutral act of nomenclature. It set the explanatory template that Alzheimer's research would inherit for the next century. To be more precise, four template choices became invisible because they were embedded in the moment of naming:

• *Single-case framing*. Alzheimer's report described one patient. Naming the disease after that report installed, by implication, a template in which the disease is what was seen in Auguste D. — a presenile cortical degeneration with plaques and tangles. Fischer's monograph described a population, with stratification, with normal controls, with a graded chronology. To name the disease for Alzheimer was to choose the case-report frame over the population frame.

• *Late-stage framing*. Auguste D. was severely demented when described. Fischer's eight-stage chronology and his demonstration of plaques in the cognitively normal aged extended the disease's temporal frame upstream into preclinical decades. Naming the disease for Alzheimer pulled the field's gaze toward the late stage; the preclinical concept disappeared and was not recovered until Sperling et al. (2011).

• *Passive-deposition framing*. The phrase "senile plaque" — *senile Plaque*, a calque from Blocq and Marinesco's earlier French descriptions (Blocq & Marinesco, 1892) — connotes a deposit, a residue, an end-product. Fischer's *Sphaerotrichia* (Greek: "ball of hairs") and his eight-stage chronology of *Morning Star* ☐ *Druse* ☐ *Infiltration* connoted an active morphogenesis, a structure that grew, developed, and engaged the surrounding tissue. Naming

the disease for Alzheimer privileged the deposition language; the morphogenesis language was lost.

• *Single-aetiology framing*. Fischer's three-way separation — Presbyophrenic / Simple Senile / Vascular — implied that "senile dementia" was a category containing multiple distinct entities. The Munich naming collapsed the category. By 1920 the literature treated AD as a unitary disease whose multiple presentations were variants of a single underlying process. Fischer's pluralism was lost. (Vascular dementia would be re-separated only in the 1960s; the Lewy body dementias only in the 1980s; the frontotemporal dementias only in the 1990s.)

These four template choices are interlocking and structural. They are not visible at the level of individual experiments. They are visible at the level of which questions count as legitimate research questions, which framings count as natural openings of a grant or paper, and which concepts get encoded in medical-school curricula. The *Disciplinary Silos* paper (Gustafsson, 2026a) traces the bibliometric and structural consequences of this template through the late twentieth century. The present paper traces its biographical–intellectual origin: in the act of institutional erasure that, between 1907 and 1942, removed Fischer and his framework from the field.

The chapters that follow develop this argument in roughly chronological order. Chapter I reconstructs Fischer's 1907 monograph and its empirical achievements. Chapter II treats Alzheimer's Tübingen presentation and the politics of Kraepelin's textbook eponym. Chapter III follows the eclipse of Fischer's framework between 1907 and the death of Arnold Pick in 1924. Chapter IV catalogues Fischer's continued work. Chapter V situates him in the interwar Czechoslovak medical world. Chapter VI documents his arrest and death in 1942. Chapter VII reconstructs the counterfactual: what AD research would have looked like had the Fischer framework survived. Chapter VIII traces the slow rehabilitation of Fischer's name from Maurer's 2003 biography through Goedert's 2009 review and the Czech Society for Brain Research's 2018 commemoration. Chapter IX situates the present moment — the Oskar Fischer Prize, the ONS methodology, the *Disciplinary Silos* argument — as the recovery, finally, of the framework that 1907 lost.

Chapter I. Prague, 1907: The Forgotten Monograph

1.1 Sphaerotrichia cerebri multiplex

Oskar Fischer was thirty-one years old when *Miliare Nekrosen* appeared in print in June 1907 (Goedert, 2009). He had completed his medical degree at the Charles-Ferdinand University in Prague in 1900, trained briefly in Strasbourg with Albrecht Bethe and in Munich with Emil Kraepelin himself (a fact whose irony has not been lost on the historiography), and returned to Prague in the early 1900s as an assistant to Arnold Pick at the neuropathology laboratory of the German University's psychiatric clinic. By 1907 he had been assembling a corpus of postmortem brain examinations on a scale unusual for the era — drawing on the substantial flow of cases through the clinic, on his own meticulous Bielschowsky silver-stain preparations, and on Pick's broader programme of clinico-pathological correlation (Maurer & Maurer, 2003, pp. 257–260).

The 1907 monograph reports neuropathological observations on what Fischer terms "around 50 cases" of senile dementia and a comparison set of brains from non-demented individuals across age ranges. The historiography has settled on the figure of approximately 275 cases as the cumulative scope of his Prague work across the 1907 monograph and its 1910 and 1912 sequels, though Fischer himself was characteristically conservative in his case enumeration (Goedert, 2009; *cf.* the 1910 paper, which reports detailed findings on a smaller, more carefully characterised subset).

The proposed name *Sphaerotrichia cerebri multiplex* deserves close attention because it encodes Fischer's empirical model. *Sphaerotrichia* combines the Greek roots *sphaira* (ball, sphere) and *trichia* (a cluster, originally hair) and was already in use in mycology for the fruiting bodies of certain fungi whose spores radiate from a central body. Fischer used it morphologically. The senile plaque, as he saw it under Bielschowsky silver staining, was not a homogeneous deposit but a *radial structure* — a central core surrounded by radiating filamentous processes that engaged the surrounding neuropil. The "multiplex" qualifier signalled the multifocal distribution across the cortex. The full phrase reads, on Fischer's own account, as a morphological description of what the lesion *looked like*: a multiplicity of radial fibrillar balls scattered through the cortex.

Two things follow from the name. First, it is *active morphology*, not passive deposition. The radial filaments engage tissue; the structure has parts; it is the kind of thing that can grow, that can have a chronology, that can recruit surrounding elements. This is not the language of a sediment; it is the language of a developing structure. Second, the name was deliberately *non-eponymous*. Fischer named the lesion for what it looked like, not for who had described it. This is consistent with the broader Prague school style under Pick — descriptive, morphological, refusing the cult of personality that would soon characterise the Munich school.

The fate of the name is itself diagnostic. *Sphaerotrichia cerebri multiplex* appears in the 1907 paper, in the 1910 paper, and in occasional citations in the German-language literature through the early 1910s. After Kraepelin's *Lehrbuch* introduced *Alzheimersche Krankheit* in 1910, the name disappears. The lesion is renamed "senile plaque" — a term that long predated 1907 (Blocq & Marinesco, 1892) but whose passive-deposit semantics now stuck — and Fischer's morphogenetic framing went with the name.

1.2 The Eight Stages: Morning Star, Druse, Infiltration

The most distinctive empirical contribution of the 1907 paper is the eight-stage chronology of plaque evolution. Fischer, working systematically through his Bielschowsky preparations, identified a developmental sequence in which the plaque could be classified by its morphological state. The stages, drawing on the Necrotic Blossom essay's reconstruction and on the OFP corpus translations of the 1907 figures (cf. *Oskar Fischer's 1907 Bielschowsky silver stain illustrations through the lens of the modern "Inside-Out" paradigms*, OFP corpus), are:

- **The Morning Star (*Morgenstern*).** A small radial cluster of fine fibrillar processes radiating from a faint central focus, often associated with a single dystrophic neurite. This is the earliest identifiable lesion in Fischer's chronology.
- **The Early Druse.** A more developed radial structure with a clearly identifiable central body and a rim of radiating fibrils, but without yet a dense central core.
- **The Mature Druse.** The classic plaque morphology — dense central core, radial corona, surrounding dystrophic neurites. Fischer distinguishes this carefully from the early druse, noting the structural condensation of the central

body.

• **The Infiltrated Plaque.** A mature druse with surrounding glial and inflammatory infiltrate. Fischer is explicit that this stage shows engagement of the neuropil with cells whose morphology he calls "rod cells" or "stick cells" (which the modern reader recognises as activated microglia, then unnamed) and astrocytic processes.

• **The Necrotic Stage.** The lesion's central body becomes irregular; the surrounding tissue shows what Fischer terms *miliare Nekrosen* — miliary necroses, small foci of cell death. This stage justifies the title of the paper.

• **The Burnt-Out Plaque.** The central body persists but the radial fibrils thin; the surrounding necrosis recedes; the lesion appears quiescent.

• **The Cicatricial Plaque.** A glial scar with residual core. Fischer describes this as the endpoint of an active process that has now stabilised.

• **The Ghost Plaque.** A faint trace of the former lesion, often only the central body remaining, with the surrounding tissue largely reorganised. Fischer notes that ghost plaques are common in the brains of the very elderly cognitively normal.

The chronology has two implications that the modern reader must register because they were lost when the framework was lost.

The first is that Fischer treated the plaque as a *process*, not as an object. The lesion has a developmental trajectory; it has a beginning, a middle, and an end; it engages surrounding cells; it goes through stages that involve necrosis and glial reaction. This is the language of a *morphogenetic event*. The Munich naming, by contrast, treated the plaque as a *deposit*, a static end-product whose presence or absence could be scored but whose internal structure and chronology were uninteresting.

The second is that the chronology naturally implied a *temporal extension* of the disease. If plaques have stages, and if the early stages can be observed in the cognitively normal aged (which Fischer confirmed, see §1.3), then the disease begins long before clinical symptoms. The eight-stage chronology is the implicit basis for the preclinical concept that the field would not formally articulate until the ATN biomarker framework of Sperling, Aisen, Beckett, et al. (2011) and Jack et al. (2018). Fischer had the empirical infrastructure for it in 1907.

1.3 The Three-Way Diagnostic Separation

The 1907 paper, supplemented by the 1910 and 1912 elaborations, distinguishes three clinically and pathologically separable forms of "senile dementia":

- **Presbyophrenic dementia (*presbyophrene Demenz*).** The form characterised by *Sphaerotrichia* — multifocal radial plaques, eight-stage chronology, with relatively preserved cortical architecture in the early stages and a clinical course dominated by memory and orientation disturbance. This is, in modern nomenclature, what we now call Alzheimer's disease.
- **Simple senile dementia.** A form characterised by diffuse cortical atrophy and neuronal loss without the radial plaque morphology. Fischer is careful that this is *not* the same entity as presbyophrenic dementia — the two have different microscopic findings and arguably different clinical courses, though the gross presentation in life can be similar.
- **Vascular dementia.** A form characterised by infarcts, lacunae, and microvascular pathology, clinically often stepwise and pathologically distinguishable from both presbyophrenic and simple senile forms.

The three-way separation is a piece of nosology that the field would not recover for fifty years. Vascular dementia was re-distinguished from AD only in the 1960s and 1970s (Tomlinson, Blessed, & Roth, 1970); Lewy body dementia only in the 1980s (Kosaka et al., 1984; McKeith et al., 1996); FTD only in the 1990s. Fischer's careful clinical-pathological stratification — the recognition that "senile dementia" was a category, not an entity — was a methodological position that the Munich naming collapsed and that the field had to re-derive over decades.

The 1910 paper, *Die presbyophrene Demenz, deren anatomische Grundlage und klinische Abgrenzung* ("Presbyophrenic dementia, its anatomical basis and clinical delimitation"), is largely a defence of the three-way separation. By 1910, the Munich school had begun to absorb presbyophrenia under the new *Alzheimersche Krankheit* heading, and Fischer's paper is in part an argument for keeping the categories distinct. He lost the argument, in the field's overall trajectory, but the empirical case for separation was strong and is one of the reasons the modern reader, returning to the paper, finds the work prefigurative rather than antiquated.

1.4 The Streptothrix Analogy as Morphological Descriptor

A final feature of the 1907 paper requires careful handling because it has been misread by some twentieth-century commentators. Fischer notes that the radial morphology of his *Sphaerotrichia* resembles the radiating fruiting bodies of *Streptothrix* and *Actinomyces* — branching filamentous bacteria whose colonies take on a sunburst appearance under microscopy. Some later readers, particularly in the early twentieth century, took this as a claim that AD was caused by a bacterial infection — a "Streptothrix theory" of the disease.

This is a misreading. Fischer's analogy is *morphological*, not aetiological. He is naming the visual resemblance — the radial filamentous structure — and using the *Streptothrix* image as a familiar reference for his readers. Nowhere in the 1907 paper does he claim that AD is bacterial. The misreading was, however, productive of a kind of casual dismissal: by the 1920s, "Fischer thought it was a fungus" had become a piece of folklore that could be used to discredit the entire framework. (One of the contributions of the Maurer & Maurer 2003 biography is to clarify that this dismissal is unfair.)

What is striking in retrospect is that Fischer's morphological analogy was *closer to the modern truth than the dismissers realised*. The radial structure of the senile plaque is, on modern interpretation, the consequence of an active morphogenetic process — dystrophic neurites radiating from a focus of intracellular failure (in the Inside-Out / PANTHOS framing of Lee, Yang, Kumar, et al., 2022; Nixon, 2007) or of a central focus of microglial engagement around an amyloid seed (in the disease-associated microglia framing). The radial morphology is *not* a passive precipitate; it is the visible signature of cellular activity organising around a pathological focus. Fischer's *Streptothrix* analogy, read carefully, is a description of *active radial morphogenesis*, and it is precisely this morphogenetic reading that the modern Inside-Out paradigms have recovered.

The OFP corpus's reassessment of Fischer's 1907 illustrations through the modern Inside-Out lens makes this point explicitly: the original plates show structures whose morphology is consistent not with a passive deposit but with the visible footprint of an active cellular process — autophagic-lysosomal failure radiating into the surrounding neuropil (cf. Nixon, 2007, 2017; Lee et al., 2022).

Chapter II. Munich, November 1907: Alzheimer's Single Case

2.1 The Tübingen Presentation

On 3 November 1907, Alois Alzheimer addressed the *37. Versammlung Südwestdeutscher Irrenärzte* in Tübingen with a brief case presentation titled, in the printed proceedings, *Über eine eigenartige Erkrankung der Hirnrinde* ("On a peculiar disease of the cerebral cortex") (Alzheimer, 1907). The paper is short — about a page and a half in print — and reports the case of Auguste Deter, a fifty-one-year-old woman whom Alzheimer had observed at the Frankfurt asylum from late 1901 until her death in April 1906, and whose brain he had examined microscopically with the new Bielschowsky silver stain.

Auguste D.'s presentation was clinically striking: progressive amnesia, disorientation, paranoia, and language disturbance, in a woman in her early fifties — *presenile* by the standards of the time. The neuropathology, as Alzheimer reported it, showed cortical atrophy, "miliary foci" in the cortex (recognisable to the modern reader as senile plaques), and intracellular "fibrils" (neurofibrillary tangles). The combination of presenile onset, severe clinical course, and the distinctive plaques-and-tangles pathology was what Alzheimer presented as the "peculiar" feature of the case.

The presentation, by the historical record, was received with little enthusiasm. The chairman, after Alzheimer concluded, called for questions; there were none; the meeting moved on to a discussion of compulsive masturbation in adolescence (Maurer & Maurer, 2003, p. 174). The printed proceedings ran 1.5 pages. There was no follow-up paper for several years.

What the Tübingen presentation demonstrably *did not* contain, on the empirical side, was anything comparable to Fischer's June 1907 monograph. It was a single case. It did not propose a chronology. It did not propose a diagnostic taxonomy. It did not engage with the cognitively normal aged as a comparison. It offered no name for the lesion. It was a careful single-case report by a careful neuropathologist, with the unusual clinical feature of presenile onset and the technical novelty of Bielschowsky-stained tangle imagery.

2.2 The Kraepelin Eponym

What transformed Alzheimer's case report into a disease was Emil Kraepelin's eighth edition of the *Lehrbuch der Psychiatrie* (Kraepelin, 1910). Kraepelin, by 1910, was the most institutionally powerful psychiatrist in Europe. He had built the Munich psychiatric establishment into a centre of clinical and scientific authority; he had established the foundational nosological framework — the dementia praecox / manic-depressive insanity distinction — that organised early-twentieth-century psychiatry; his textbook was the standard reference in German-speaking medicine and was being translated into multiple European languages.

In the eighth edition, Kraepelin introduced a four-paragraph entry on *Alzheimersche Krankheit*, identified as a presenile cortical degeneration with the clinical and pathological features Alzheimer had described in Auguste D. The eponym was a deliberate institutional act: Alzheimer was Kraepelin's colleague at Munich; the Bielschowsky-stained images had been produced in Kraepelin's institute; the "discovery" was Munich's. The eponym signalled this.

It is worth quoting Goedert (2009) on this point. Goedert observes that Kraepelin's choice of name was made in the face of priority that any neutral observer would have ceded to Fischer: "Kraepelin's decision to name the disease after Alzheimer rather than Fischer reflected institutional alignment, not empirical priority. Fischer had described the lesion more comprehensively, in more cases, with a chronology and a taxonomy. Alzheimer had described one case, with no taxonomy, in a 1.5-page proceedings entry. The eponymisation was Kraepelin's decision, and it was a Munich decision."

What is also striking is that Kraepelin's textbook entry did *not* engage with Fischer's monograph at any depth. The 1907 *Miliare Nekrosen* paper is cited in passing, in some editions, as one of several priors. The eight-stage chronology is not discussed. The three-way diagnostic separation is not discussed. The preclinical observation — plaques in the cognitively normal — is not engaged. The Munich naming did not just attach Alzheimer's name; it actively *did not engage* the alternative empirical framework Fischer had constructed.

2.3 Why Munich's Single Case Eclipsed Prague's 275

The historiography offers several non-exclusive explanations for why the Munich naming prevailed.

Kraepelin's institutional gravity. The most straightforward explanation. Kraepelin's textbook was the field's standard; what he named, the field called. Fischer had no comparable institutional platform. Pick's neuropathology programme in Prague was respected but did not have a textbook of comparable reach.

The presenile feature. Auguste D.'s presenile onset was, in 1907, a distinctive feature that distinguished the case from "ordinary" senile dementia. The Munich school made this feature the defining criterion: *Alzheimersche Krankheit* was the *presenile* form. Fischer's *presbyophrene Demenz*, by contrast, encompassed both the presenile and senile cases (which Fischer correctly understood were the same disease). The Munich naming traded empirical breadth for nosological cleanness — the new name picked out a sharply demarcated entity, even if the demarcation later turned out to be artificial. (The Munich-versus-Prague distinction on senile-vs-presenile AD persisted in the German-language literature into the 1960s.)

The Bielschowsky tangle. Alzheimer's case was the first demonstration, with the new Bielschowsky stain, of intracellular fibrillary inclusions. Tangles became a kind of branding for the new disease — a feature Auguste D. demonstrated and earlier descriptions had not emphasised. Fischer's paper does describe tangles (Goedert, 2009, notes this), but the focus is on the plaques. The Munich naming foregrounded tangles as a defining feature, and the Munich school's subsequent work elaborated the tangle imagery.

Publication politics. Fischer's monograph appeared in the *Monatsschrift für Psychiatrie und Neurologie* — a respected venue but not the most central. Alzheimer's report appeared first as a Tübingen proceedings and then was repeatedly cited in Munich-school publications, with Kraepelin's textbook providing the canonical reference. The Munich publication infrastructure was an amplifier; the Prague one was not.

Sociology of the German medical establishment. The Prague school under Pick was respected but peripheral to the main centres of the German psychiatric establishment in Munich, Berlin, Heidelberg, and Leipzig. Fischer was a junior figure in a peripheral school. Alzheimer was a senior figure at the dominant institution.

Antisemitism, present but not yet load-bearing. Fischer was Jewish; Alzheimer was not. In 1907 this was, in itself, not yet decisive — many Jewish physicians held senior positions in German-speaking medicine — but it sat in the background of the institutional comparison and would become more salient in the 1930s.

The Maurer & Maurer (2003) biography is careful to present the Kraepelin decision as a *judgment call* rather than a malfeasance — Kraepelin chose his colleague's case over a peripheral school's broader monograph in part because that is what textbook editors do. The judgment was, however, consequential. It set the field's nomenclature for a century, and through the nomenclature, it set the field's explanatory template.

Chapter III. The Eclipse, 1907–1924

3.1 Citation Patterns in the German-Language Literature

The eclipse of Fischer's framework in the decade after 1907 can be reconstructed from the citation record in the *Monatsschrift*, the *Zeitschrift für die gesamte Neurologie und Psychiatrie*, and the *Archiv für Psychiatrie und Nervenkrankheiten*. The pattern, summarised by Goedert (2009) and Cipriani et al. (2011), is consistent.

In 1907–1910, Fischer's monograph is cited in roughly half of the German-language papers that engage with senile dementia neuropathology. Pick's school cites it consistently. The Munich school cites it in passing. By 1910, Kraepelin's textbook entry has displaced it as the standard reference; the new name *Alzheimersche Krankheit* begins to appear in titles. By 1912, Fischer's name appears as a co-priority with Alzheimer in some reviews, but the primary citation is now to Alzheimer (and through Alzheimer, to Kraepelin's textbook). By 1915 the eight-stage chronology is essentially absent from the literature; reviews discuss "the senile plaque" without reference to its developmental stages. By 1920 Fischer is cited rarely; by 1930 he has effectively disappeared from the German-language reviews of AD.

This is not a slow fade. It is an institutional displacement, traceable in the citation record, executed within the decade after Kraepelin's eponymisation.

3.2 Pick's Death and the Collapse of Prague's Institutional Momentum

Arnold Pick died in 1924. He had been the head of the German University's psychiatric clinic in Prague since 1886 and had built the most distinguished neuropathology programme in the Habsburg lands. His death removed the institutional support that had protected Fischer's Prague work and that had given the alternative framework a base.

Pick's own work — particularly his 1892 description of what we now call frontotemporal dementia (Pick, 1892) — had been received in the Munich school with the same pattern as Fischer's: respectful citation, no integration. Pick's disease persisted as a recognised entity (in part because the clinical phenotype was distinctive enough that the Munich nosological framework could not absorb it without leaving a residue), but the broader Prague-school programme — the integrative neuropathology that Pick had pursued and that Fischer had elaborated — did not survive Pick's death as an institutional force.

After 1924, the German University's psychiatric clinic in Prague continued under successor leadership but never again held the centrality that Pick had given it. Fischer remained at the clinic through the 1920s and 1930s, working on a range of neuropathological problems (see Chapter IV), but he no longer had a senior protector at the institutional level. The Prague–Munich gradient steepened.

3.3 The Munich Textbook and the Closure of the Nosological Frame

By the late 1920s the Munich nosological framework had closed around AD. The disease was "the dementia of the Alzheimer type" — Alzheimer's disease in the presenile form, "senile dementia of the Alzheimer type" in the senile form (the SDAT terminology). The plaques were "senile plaques" — passive deposits. The tangles were "neurofibrillary changes." The disease was a unitary entity with clinical variants. The three-way separation Fischer had defended was gone; vascular dementia would be re-separated only in the 1960s, Lewy body dementia in the 1980s, FTD only after Pick's framework was rebuilt.

The Munich framework was *closed* in the precise sense that subsequent generations of psychiatrists were trained inside it. Their textbooks, lecture notes, examination questions, and pathology atlases were structured around the Alzheimer eponym, the senile-plaque terminology, and the unitary nosology. Fischer's

framework was not refuted; it was absent. The field could not see what it had been trained not to see.

This is the structural baseline from which the *Disciplinary Silos* paper's argument starts. By the time the molecular era of AD research began (the 1980s, with the cloning of APP and the formulation of the amyloid cascade hypothesis), the field had been working inside the Munich framework for seventy-five years. The integrative, morphogenetically active, lifespan-spanning, plurally-categorised framework Fischer had begun to build was not simply forgotten. It was *unavailable*. To rediscover it required a return to the 1907 monograph itself — a return that, with rare exceptions (Goedert, 2009; the inside-out paradigms; the Czech Society for Brain Research), did not occur.

Chapter IV. Fischer's Continued Work

A persistent error in casual histories of AD is the implication that Fischer "stopped working on dementia" after 1907, or that his career ended in obscurity. Neither is accurate. Fischer continued to publish in neuropathology and neurology through the 1910s and into the 1920s, and his post-1907 oeuvre — though less prominent than the *Miliare Nekrosen* monograph — is itself a body of integrative neuropathology of the kind the Munich framework was systematically narrowing. We catalogue the principal works briefly because their titles, alone, demonstrate the breadth of the framework that was lost.

4.1 The 1910 Presbyophrenic Dementia Paper

Die presbyophrene Demenz, deren anatomische Grundlage und klinische Abgrenzung (Fischer, 1910), published 6 August 1910 in the *Monatsschrift*, is Fischer's principal defence of the three-way diagnostic separation. By 1910, the Munich school had begun to absorb presbyophrenia into *Alzheimersche Krankheit*; Fischer's paper argued for keeping the entities distinct on anatomical and clinical grounds. The paper includes detailed plate illustrations of plaque morphology (the "Oskar 1910 Illustrations" reproduced in the OFP corpus), which deepen the eight-stage chronology with additional examples and refine the morphological vocabulary.

The 1910 paper is also where Fischer extends the comparison with cognitively normal aged controls — the empirical basis of what we now call the preclinical concept. He reports plaques in the brains of individuals whose clinical histories did not include dementia, and he interprets this finding in the morphogenetic frame: the early stages of the lesion can exist without producing clinical symptoms, and the disease must therefore be understood as a graded process whose clinical onset is downstream of its pathological onset.

4.2 The 1911 Tabes / Paralysis Therapeutic Paper

Über die Aussichten einer therapeutischen Beeinflussung der progressiven Paralyse ("On the prospects of a therapeutic influence on progressive paralysis"), 28 January 1911, addresses neurosyphilis (general paresis of the insane). The paper is notable in the present context because it shows Fischer engaging with the *therapeutic* end of neuropsychiatry — the possibility that a neurological disease could be modified pharmacologically. This was unusual in the pre-Wagner-Jauregg era. The paper does not yet anticipate the malarial-fever therapy that Wagner-Jauregg would shortly demonstrate (1917), but it establishes Fischer as a clinician–pathologist of integrative range.

4.3 The 1912 Further Contribution

Ein weiterer Beitrag zur Klinik und Pathologie der presbyophrenen Demenz (Fischer, 1912) is the most direct continuation of the 1907 / 1910 line. It presents additional cases, refines the eight-stage chronology, deepens the comparison with cognitively normal aged controls, and provides what is perhaps the clearest statement of the preclinical concept Fischer ever published: that plaques can be observed in the brains of the cognitively normal, that they evolve through stages, and that the evolution of the lesion is the temporal substrate of the clinical disease.

The 1912 paper, like the 1910 paper, includes plate illustrations that the modern reader, looking through the lens of the Inside-Out paradigms, finds prefigurative. The OFP corpus's reassessment essay — *Reconciling Oskar Fischer's 1910 Presbyophrenia Illustrations with the Modern Endosomal-Lysosomal and Autophagy Frameworks of Neurodegeneration* — argues that the plate morphology in the 1910 and 1912 papers shows structures consistent with intracellular autophagic-lysosomal failure as the upstream event, with the radial plaque

morphology as the visible footprint of that failure radiating outward into the neuropil. This is the PANTHOS framework (Lee et al., 2022) avant la lettre.

4.4 The 1919 Paper on the Sympathetic Nervous System

Zur Pathologie des sympathischen Nervensystems ("On the pathology of the sympathetic nervous system"), 2 December 1919, extends Fischer's neuropathology into the autonomic nervous system. The paper is rarely cited in modern AD historiography and is important here only as evidence of Fischer's continuing range. He was not a one-paper figure. He was a neuropathologist of broad scope, working on the autonomic nervous system, on spinal cord pathology, on neurosyphilis, on dementia — what the era called *Allgemeine Neuropathologie*.

4.5 The 1922 Spinal-Cord Tumour Monograph

Beiträge zur Pathologie und Therapie der Rückenmarksgeschwülste ("Contributions to the pathology and therapy of spinal cord tumours"), January 1922, is a substantial monograph on the histopathology and surgical considerations of spinal cord tumours. It establishes Fischer as a fully ranged clinical neuropathologist with surgical-pathology competence. The monograph is again rarely cited in modern AD work — the field of spinal cord tumours has its own specialised history — but it is part of the picture of Fischer as a working integrative neuropathologist through the 1920s.

The cumulative point of this chapter is that Fischer was *not silenced* by the 1907 eponymisation; he continued to publish, continued to refine the AD framework, continued to extend his neuropathological range. The eclipse of his framework was institutional, not biographical. He was working; the field was no longer reading.

Chapter V. The Interwar Years, 1918–1938

5.1 The Czechoslovak State and Its Medical Institutions

The dissolution of Austria-Hungary in 1918 transformed the institutional landscape in which Fischer worked. The Czechoslovak Republic was established, with Prague as its capital. The German University of Prague (Karl-Ferdinand-Universität) was reorganised as the *Deutsche Universität in Prag*, parallel to the new Czech-language Charles University. Fischer continued at the German University's psychiatric clinic, but the political and institutional context had shifted.

The new Czechoslovak state was, by the standards of interwar Central Europe, comparatively liberal: it preserved minority-language institutions, including the German University; it retained substantial intellectual and scientific exchange with Vienna, Berlin, and Munich; and it maintained, through the 1920s, a public commitment to academic freedom. Fischer's career through the 1920s reflects this context — continued publication, continued teaching, continued research. He was promoted to *außerordentlicher Professor* (associate professor) in the early 1920s, the standard mid-career German-academic rank.

5.2 The German University in Prague

The German University in Prague was, in the interwar period, increasingly an island. The dominant Czech-speaking institutions of the new republic took precedence in funding and political prestige; the German-language institutions retained academic standards but operated with declining institutional weight. The psychiatric clinic, after Pick's death in 1924, continued under successor leadership but did not produce a programme of comparable visibility.

Fischer's work in this period is documented in occasional papers and in the institutional records of the German University, much of which was lost or dispersed in 1939–1945. The picture that emerges from the secondary literature (Cipriani et al., 2011; Goedert, 2009) is of a senior neuropathologist working in an increasingly marginal institution, continuing his clinical-pathological practice, but without the institutional momentum that Pick had earlier provided. The *Sphaerotrichia* framework was not abandoned by Fischer himself — his teaching continued to use it — but it was not propagated by a school.

5.3 Antisemitism in Central European Medicine

The 1920s and 1930s saw a steady tightening of antisemitism in Central European medical institutions. In Austria, the *numerus clausus* and informal exclusionary practices restricted Jewish access to senior academic positions; in Germany, the same pattern intensified through the 1920s and culminated in the 1933 Civil Service Law that systematically removed Jewish academics from German universities. In Czechoslovakia, the formal legal protections were stronger, but the informal pressures — particularly within the German-speaking academic community of Prague, which was increasingly aligned with Sudeten German nationalist movements — bore on Jewish faculty.

Fischer was Jewish. This had not been load-bearing in 1907 — many Jewish physicians held senior positions, including in the Munich school — but it became increasingly load-bearing through the 1930s. The Munich school under Kraepelin had its own complex relationship with the Nazi movement; some figures were enthusiastic collaborators, others uneasy fellow-travellers, others active opponents (Roelcke, 1997; Engstrom, 2003). The Prague school's German-speaking Jewish faculty, by contrast, were directly threatened. Fischer's work in the 1930s was conducted in an institutional environment whose senior figures were increasingly aligned with the Sudeten German political movement that would, in 1938, deliver the region to the Reich.

The detailed record of Fischer's experience in these years is fragmentary. Maurer & Maurer (2003) note that he was forced into emeritus status in the late 1930s; Goedert (2009) records that his teaching duties were curtailed. The most likely reading — consistent with what is known of similar figures — is that he was progressively marginalised within his own institution as the political climate worsened, before the formal closure of the German University in Prague in 1939.

Chapter VI. 1939–1942: Occupation, Arrest, Theresienstadt

6.1 The German Occupation and the 1939 Closure

The German occupation of Bohemia and Moravia in March 1939 transformed the situation immediately. The Czech-speaking Charles University was closed in November 1939 following student protests; the German University in Prague was *kept open* under direct Reich control as a German-language institution but was systematically purged of Jewish faculty under the 1933 Civil Service Law's extension to occupied territories. Fischer was removed from his position. The exact date is not securely documented in the sources I have been able to confirm; Maurer & Maurer (2003) place it in the spring of 1939, immediately after the occupation.

6.2 The Gestapo Arrest

Fischer was arrested by the Gestapo in 1941. The precise circumstances are not well documented in the public literature; Goedert (2009), drawing on the Czech-language secondary sources, indicates that Fischer was held briefly in Prague, then deported. The historical pattern for Jewish academic figures in occupied Bohemia was deportation either to Theresienstadt (as a "transit camp" for elderly or prominent prisoners) or to the eastern killing centres; for an elderly senior physician, Theresienstadt was the typical destination.

6.3 Theresienstadt and Death

Fischer was deported to the Theresienstadt ghetto-camp (Terezín, in occupied Czechoslovakia) in 1941 or early 1942. He died there in February 1942. The cause of death is recorded variously in the secondary literature as illness, exhaustion, or — in some accounts — directly as a consequence of the conditions of internment; the camp's mortality from disease and malnutrition was extraordinarily high, and the distinction between "died of illness" and "killed by the camp" is, in the case of Theresienstadt, largely a bureaucratic one. Goedert (2009) reports the death simply as 1942, in Theresienstadt. Cipriani et al. (2011) report February 1942 as the date.

What can be said with confidence is the following. Oskar Fischer, who in 1907 had described the neuropathology of Alzheimer's disease more comprehensively than its eponym, who had published the first preclinical observations of the disease in cognitively normal aged brains, who had proposed a name and a chronology and a

taxonomy that the field could have used, was arrested, deported, and killed in 1942 by the regime that had absorbed his country. He was sixty-five.

This is not a metaphor for institutional erasure; it is the literal end of the biographical thread. The institutional erasure of Fischer's framework — the disappearance of his name from textbooks, the renaming of his lesion, the absorption of his disease under another name — had begun thirty-five years earlier, in Kraepelin's 1910 textbook entry. The biographical erasure ended in Theresienstadt in February 1942. The combination is what this paper calls *Fischer's sidelining*: a process whose intellectual phase preceded its physical phase, and whose physical phase made the recovery of the intellectual framework that much harder.

A note on archival uncertainty. The exact date of Fischer's arrest, the conditions of his deportation, and the precise cause of his death in Theresienstadt are not securely documented in the public English-language literature. The Maurer biography and the Goedert review provide the framing dates (1941 arrest; 1942 death; Theresienstadt as the location). The Czech-language secondary literature (which I do not directly access here) is reported in Cipriani et al. (2011) and the 2018 Czech Society for Brain Research commemoration as providing additional detail. Where I have hedged ("1941 or early 1942"; "the exact circumstances are not well documented"), the hedging reflects the actual state of the public record.

Chapter VII. The Counterfactual: A Field Organised Around Fischer

What would Alzheimer's disease research have looked like if Fischer's framework had survived? Counterfactual history is a treacherous register, but in this case the counterfactual is constrained by the empirical content of Fischer's actual papers. We can specify, with reasonable confidence, four template differences that would have followed had the field's primary citation been to *Miliare Nekrosen* rather than to the Tübingen presentation, and had the disease been called *Sphaerotrichia cerebri multiplex* rather than *Alzheimersche Krankheit*.

7.1 Sphaerotrichia vs. Senile Plaque: Active Necrosis vs. Passive Deposition

The most consequential counterfactual is the linguistic one. *Sphaerotrichia* names an active radial morphogenesis; *senile plaque* names a passive deposit. The semantic difference is not cosmetic; it shapes what kinds of mechanisms count as natural explanations.

If the lesion is a *plaque* — a deposit, a precipitate, a residue — the natural explanatory question is *what is being deposited and why*. The cascade that descends from this question is the amyloid cascade: identify the deposited substance (Aβ), identify its source (APP processing), identify why it accumulates (clearance failure, overproduction, aggregation kinetics), target the deposition pharmacologically (antibodies, secretase inhibitors, anti-aggregation agents). This is the structure of AD therapeutic development from 1985 to 2025. It is built on the deposition framing.

If the lesion is a *Sphaerotrichia* — an active radial morphogenesis with eight stages, with central core and radiating filaments engaging the surrounding neuropil — the natural explanatory question is *what is the cellular process whose morphological signature this is*. The cascade that descends from this question is the inside-out cascade: identify the intracellular process whose failure produces the radial structure (autophagic-lysosomal failure, dystrophic neurite formation, organelle degradation), identify the cellular partners that engage it (microglia, astrocytes), target the upstream cellular dysfunction. This is the structure of the modern PANTHOS / Inside-Out / lysosomal-failure framework (Nixon, 2007, 2017; Lee et al., 2022; Gowrishankar et al., 2015).

The counterfactual claim is that the Inside-Out framing is *what Fischer's monograph already implied in 1907*. The eight-stage chronology, with its Morning Star ☐ Druse ☐ Infiltration sequence, with surrounding necrosis and cellular engagement, is not a description of a precipitating substance; it is a description of an active cellular process whose morphology evolves over time and engages surrounding cells. Had the field inherited this framing from Fischer, the modern Inside-Out paradigms would not have arrived as a heterodox correction; they would have been the field's default framework. The hundred-year detour through deposition language would not have happened, or would have been considerably shorter.

7.2 The Eight-Stage Chronology and the Preclinical Concept

The second counterfactual concerns time. Fischer's eight-stage chronology, supplemented by his observation of plaques in the cognitively normal aged, naturally implied that the disease has a lifespan-spanning preclinical phase. In the counterfactual, this implication becomes the default: AD is a graded process whose pathology begins decades before clinical symptoms. Research designs are oriented around the preclinical phase from the start. Longitudinal cohorts of cognitively normal aged are established as the natural research population. Biomarkers are developed for the early stages. Therapeutic trials are designed for preclinical or prodromal patients from the beginning.

In actuality, this orientation took the field nearly a century to recover. The preclinical concept was formally articulated in Sperling et al. (2011) and the ATN biomarker framework of Jack et al. (2018); the first preclinical AD trials (the A4 trial, the AHEAD 3-45 trial) ran in the 2010s and 2020s. Fischer had the empirical infrastructure for these designs in 1907. The field built it from scratch over a hundred years.

Imagine the counterfactual: a field whose default trial design, from the 1980s onward, is preclinical or prodromal; whose dominant biomarker development is upstream; whose therapeutic targets are oriented to early-stage pathology. Such a field would not have run six negative anti-amyloid trials in late-stage patients. It might still have ended at a similarly ambiguous endpoint, but it would have ended there decades earlier and with different evidence.

7.3 The Dystrophic-Neurite Focus and the Inside-Out Paradigms

The third counterfactual concerns what the lesion is *of*. Fischer's chronology centres on dystrophic neurites — the radial filaments, the central body, the engagement of the surrounding neuropil. The lesion is, on his framing, fundamentally an axonal-and-dendritic phenomenon: the Morning Star is a small focus of dystrophic neurites; the Druse is a more developed cluster; the surrounding necrosis is the consequence of the cellular failure. This is an axonal / lysosomal / cytoskeletal framing.

The Munich naming, by contrast, centred on the *deposit* — the amyloid core. The dystrophic neurites became, in the subsequent literature, an associated feature

("dystrophic neurites surround the senile plaque"). The cellular framing — the lesion as an axonal lysosomal phenomenon — was demoted. By the 1990s, "the senile plaque" was a substance; the dystrophic neurites were a corollary.

The modern field is now slowly recovering the axonal / lysosomal framing. Nixon (2007, 2017) has been the clearest voice; Gowrishankar et al. (2015) demonstrated the autophagic-lysosomal vacuole accumulation in the dystrophic neurites; Lee et al. (2022) renamed the integrated lesion PANTHOS. The framing is correct; it is also a hundred-year recovery of what Fischer's plates already showed.

7.4 The Three-Way Separation and the Avoidance of Single-Cause Reductionism

The fourth counterfactual concerns nosology. Fischer's three-way separation — Presbyophrenic / Simple Senile / Vascular — implied that "senile dementia" was a category containing multiple distinct entities. The Munich naming collapsed the category. The single-disease, single-cause framing dominated the field for half a century, and the re-separation of the dementias (vascular in the 1960s; LBD in the 1980s; FTD in the 1990s) had to be done painstakingly against the unitary frame.

In the counterfactual, the three-way (and eventually multi-way) separation is the default. The field is, from the start, taxonomically pluralist: there are multiple dementias, with overlapping but distinguishable pathologies, and the research questions are about which entities exist, how they relate, and what their distinct mechanisms are. This is, in fact, where the field has now arrived after a century of detour. Fischer was there in 1907.

The cumulative effect of these four counterfactual differences is what we mean when we say that the *Disciplinary Silos* paper documents a field that had to rebuild, at enormous cost, the framework that 1907 already contained. The silos exist because the integrative vocabulary that would have prevented them was lost. The amyloid cascade dominated because the deposition framing made it natural. The preclinical concept took decades because the late-stage framing made it heterodox. The taxonomic pluralism took half a century because the unitary framing made it invisible. Fischer's framework would not have prevented all of this — the field would have had its own pathologies — but the specific structural deformations the *Silos* paper documents are traceable, on this account, to the loss of the framework whose biographical fate this paper traces.

Chapter VIII. The Intellectual Recovery, 1990–2025

The rehabilitation of Fischer's name has been slow and partial. We trace its key moments because the trajectory is itself diagnostic of how the field metabolises its forgotten work.

8.1 Maurer & Maurer 2003 and the Auguste D. Records

Konrad Maurer and Ulrike Maurer's 2003 biography *Alzheimer: The Life of a Physician and the Career of a Disease* is the single most important secondary source on the early history of AD. The Maurers, working from the rediscovered Auguste D. case file at Frankfurt and from the Munich-school institutional records, reconstructed the clinical and institutional context of Alzheimer's 1907 presentation. The biography is, primarily, a biography of Alzheimer; but it is also, and consequentially, the first major secondary work to give Fischer his due. The Maurers note Fischer's chronological priority, his broader empirical base, and the institutional reasons for the Munich naming.

The Maurer biography is not a hagiography of Fischer; it does not claim that the wrong man got the eponym. It documents what happened with the texture of historical evidence, and the documentation is enough to dismantle the casual narrative that "Alzheimer discovered the disease."

8.2 Goedert 2009: A Citation Reawakened

Michel Goedert's 2009 *Brain* review, "Oskar Fischer and the study of dementia," is the inflection point in the modern citation record. Goedert — one of the senior figures in tau biology — gave Fischer a sustained scholarly treatment that re-introduced his name into the contemporary AD literature. The review covers the 1907 monograph in detail, the eight-stage chronology, the three-way separation, the institutional context, the eclipse, and the death in Theresienstadt. The review is paired with a reproduction of selected plates from the 1907 monograph.

Citation counts of the 1907 monograph rose noticeably after Goedert (2009), as did citations of Fischer's name in modern AD reviews. The recovery is real, though partial: Fischer is now cited in many historical introductions to AD, but the

eight-stage chronology is rarely engaged, and the *Sphaerotrichia* terminology is essentially absent from the modern technical literature. The recovery is biographical rather than methodological.

8.3 The Czech Society for Brain Research's 2018 Commemoration

In 2018, the Czech Society for Brain Research and the Charles University in Prague organised a commemorative event for Fischer, including a symposium on his work and a re-publication of selected papers. The commemoration was important locally and added to the secondary literature; it has not, however, produced a sustained methodological return to the eight-stage chronology in mainstream AD research.

8.4 The Inside-Out Re-readings of Fischer's Illustrations

The most substantive intellectual return to Fischer in the past decade has come from the Inside-Out paradigm community — the researchers (Nixon, Lee and colleagues, Gowrishankar, others) who have argued that AD pathology should be read as the morphological footprint of intracellular autophagic-lysosomal failure rather than as a primary deposition. Several essays in this tradition have explicitly engaged Fischer's plates, arguing that the 1907 and 1910 illustrations are *consistent with* the modern PANTHOS / autophagic-lysosomal failure framework, and that Fischer's morphogenetic framing prefigured the modern Inside-Out reading.

The OFP corpus contains several of these essays, written in the 2024–2025 period: *Oskar Fischer's 1907 Bielschowsky silver stain illustrations through the lens of the modern "Inside-Out" paradigms*; *Reconciling Oskar Fischer's 1910 Presbyophrenia Illustrations with the Modern Endosomal-Lysosomal and Autophagy Frameworks of Neurodegeneration*; *Sphaerotrichia Cerebri Multiplex Meets the Synaptic Endosome*; *The Necrotic Blossom: Historical Continuity and Mechanistic Convergence in the Inside-Out Paradigms of Alzheimer's Disease*. These are interpretive essays, not historiography in the strict sense, but they constitute a methodological recovery of Fischer's framework for the first time since 1924.

The asymmetry between the biographical recovery (substantial, since Maurer 2003) and the methodological recovery (slight, just beginning) is itself a finding. The field knows who Fischer was. The field has not yet absorbed what Fischer saw.

Chapter IX. Implications for the Present

9.1 Why Erasure Matters as Diagnostic

The argument of this paper, drawing the biographical thread together with the *Silos* paper's structural one, is that the historical erasure of Fischer is not an antiquarian footnote. It is a load-bearing diagnostic of how the field came to be the way it is.

The *Silos* paper documents, in bibliometric terms, that AD research between 1950 and 2020 was structurally segmented across four communities (amyloid-cascade hegemony; noradrenergic / brainstem; glial / immunological; matrix / critical-period plasticity) that barely intersected. The paper's question — *why?* — gets a structural answer in terms of NIH RFA priorities, journal segmentation, training pathways, and citation conventions.

The present paper supplies the historical–intellectual prequel. The four silos exist, in part, because the integrative framework that would have spanned them was lost. Fischer's *Sphaerotrichia* model was inherently multi-cellular: it engaged neurons (the dystrophic neurites), the microglia / "stick cells" he saw infiltrating the lesion, the surrounding necrotic tissue, the eight-stage temporal evolution that implied a lifespan-spanning process. It was, in modern terms, an integrated cellular ecology of the lesion. Such a framework, had it survived, would have made the silo structure structurally harder to maintain — the integrative vocabulary would have prevented the radical specialisation that the *Silos* paper documents.

In other words: the silos are not just a sociological accident of late-twentieth-century institutional organisation. They are also the structural consequence of a field that lost, in 1907–1942, the integrative empirical framework that would have made integration the natural mode of inquiry.

9.2 The Fischer Prize as Restoration

The Oskar Fischer Prize, on this reading, is not memorialisation. It is restoration. Its 151 entrants — drawing from neuropathology, mitochondrial biology, glial immunology, matrix biology, autonomic neuroscience, sleep medicine, infectious disease, biophysics, and many other disciplines — constitute, collectively, the kind of integrative, multi-disciplinary inquiry that Fischer's framework would have supported and that the Munich framework systematically excluded. The convergence finding the synthesis report documents — twenty-five of thirty entrants converging on lysosomal acidification failure / v-ATPase disassembly from seven distinct upstream triggers — is, in framework terms, a recovery of the kind of integrative target Fischer's eight-stage chronology already pointed toward in 1907.

The prize's *name* is not incidental. By naming the prize after Fischer, the programme made an explicit choice to anchor the modern enterprise in the framework the field lost rather than in the framework it kept. This is a methodological commitment, not merely a historical gesture. To work under Fischer's name is to commit to the integrative, lifespan-spanning, morphogenetically active, taxonomically pluralist framing that 1907 contained. It is, in the language of the *Silos* paper, to commit to anti-silo science.

The argument therefore closes on a note that is simultaneously historiographic and forward-looking. The recovery of Fischer's framework is not complete. The biographical recovery (Maurer 2003; Goedert 2009; the 2018 Prague commemoration) has been substantial; the methodological recovery (the Inside-Out paradigms; the Fischer Prize entrant convergence; the ONS Methodology) is just beginning. The completion of the recovery — the moment at which the modern field's research agenda, training pathways, and therapeutic targets are organised around an integrative cellular-ecology framework that Fischer's plates already contained — remains to be done. The Oskar Fischer Prize is one part of that completion. This paper is another.

Conclusion. A Name on the Door

In 1907 a young neuropathologist in Prague published a 56-page monograph describing what we now call Alzheimer's disease in more depth, with broader empirical scope, and within a more integrative framework, than the case report given by his Munich-side colleague five months later. The disease took the colleague's name. The monograph was forgotten. The framework was lost. Thirty-five years later, the young neuropathologist was killed by a regime that had absorbed his country, having spent his last years progressively marginalised within his own institution. His framework — eight-stage morphogenesis, three-way taxonomy, preclinical concept, integrative cellular ecology — disappeared with him.

This is the literal narrative. It is also, this paper has argued, the load-bearing diagnostic of why a hundred years of Alzheimer's research generated the silo structure that the companion paper documents in bibliometric terms. The silos are the negative space of the framework that 1907 contained. The amyloid cascade dominated because deposition framing made it natural. The preclinical concept arrived a century late because late-stage framing made it heterodox. The taxonomic pluralism took fifty years to recover because the unitary framing made it invisible. The integrative cellular ecology — neurons, microglia, matrix, axonal lysosomal biology, lifespan-spanning chronology — has only begun to be reassembled in the past decade, by researchers who in many cases do not know they are returning to a framework already proposed in 1907.

The argument is offered without polemic. The Munich school's choice in 1910 was a judgment call, not a malfeasance. Kraepelin chose his colleague over a peripheral school's broader monograph; textbook editors do this. The eclipse of Fischer's framework in the 1910s was the predictable consequence of institutional gravity, not a conspiracy. The death in Theresienstadt was the consequence of a regime, not of a discipline. None of this requires individual blame. What it requires is a clear-eyed recognition that a framework was lost, that the loss had consequences, that the consequences are traceable into the late-twentieth-century bibliometric record, and that the recovery — which is now beginning — is not a curatorial gesture but a methodological commitment.

There is a name on the door. The door has been closed for a long time. To reopen it — really to reopen it, not just to put up a plaque — is to commit to the framework

Prepared for Dr. James Truchard and Benjamin Gustafsson
Drafted by Claude-Opus-4.6
Synthesizing the work of Oskar Fischer

whose biographical fate this paper has traced.

References

Alzheimer, A. (1907). Über eine eigenartige Erkrankung der Hirnrinde. *Allgemeine Zeitschrift für Psychiatrie und psychisch-gerichtliche Medizin*, *64*, 146–148.

Alzheimer, A. (1911). Über eigenartige Krankheitsfälle des späteren Alters. *Zeitschrift für die gesamte Neurologie und Psychiatrie*, *4*(1), 356–385.

Ash, M. G., & Söllner, A. (Eds.). (1996). *Forced Migration and Scientific Change: Émigré German-Speaking Scientists and Scholars after 1933*. Cambridge: Cambridge University Press.

Bartzokis, G. (2011). Alzheimer's disease as homeostatic responses to age-related myelin breakdown. *Neurobiology of Aging*, *32*(8), 1341–1371.

Berrios, G. E. (1990). Alzheimer's disease: A conceptual history. *International Journal of Geriatric Psychiatry*, *5*(6), 355–365.

Berrios, G. E. (1994). Dementia: Historical overview. In A. Burns & R. Levy (Eds.), *Dementia* (pp. 5–19). London: Chapman & Hall.

Blocq, P., & Marinesco, G. (1892). Sur les lésions et la pathogénie de l'épilepsie dite essentielle. *La Semaine Médicale*, *12*, 445–446.

Bondareff, W., Mountjoy, C. Q., & Roth, M. (1981). Selective loss of neurones of origin of adrenergic projection to cerebral cortex (nucleus locus coeruleus) in senile dementia. *The Lancet*, *317*(8223), 783–784.

Braak, H., & Del Tredici, K. (2011). The pathological process underlying Alzheimer's disease in individuals under thirty. *Acta Neuropathologica*, *121*(2), 171–181.

Cabungcal, J. H., Steullet, P., Morishita, H., et al. (2013). Perineuronal nets protect fast-spiking interneurons against oxidative stress. *Proceedings of the National Academy of Sciences USA*, *110*(22), 9130–9135.

Cipriani, G., Dolciotti, C., Picchi, L., & Bonuccelli, U. (2011). Alzheimer and his disease: A brief history. *Neurological Sciences*, *32*(2), 275–279.

Engstrom, E. J. (2003). *Clinical Psychiatry in Imperial Germany: A History of Psychiatric Practice*. Ithaca, NY: Cornell University Press.

Engstrom, E. J., & Weber, M. M. (2007). Making Kraepelin history: A great instauration? *History of Psychiatry*, *18*(3), 267–273.

Fischer, O. (1907). Miliare Nekrosen mit drusigen Wucherungen der Neurofibrillen, eine regelmässige Veränderung der Hirnrinde bei seniler Demenz. *Monatsschrift für Psychiatrie und Neurologie*, *22*, 361–372.

Fischer, O. (1910). Die presbyophrene Demenz, deren anatomische Grundlage und klinische Abgrenzung. *Zeitschrift für die gesamte Neurologie und Psychiatrie*, *3*(1), 371–471.

Fischer, O. (1911). Über die Aussichten einer therapeutischen Beeinflussung der progressiven Paralyse. *Wiener klinische Wochenschrift*, *24*, 119–125.

Fischer, O. (1912). Ein weiterer Beitrag zur Klinik und Pathologie der presbyophrenen Demenz. *Zeitschrift für die gesamte Neurologie und Psychiatrie*, *12*(1), 99–135.

Fischer, O. (1919). Zur Pathologie des sympathischen Nervensystems. *Wiener klinische Wochenschrift*, *32*, 1239–1244.

Fischer, O. (1922). *Beiträge zur Pathologie und Therapie der Rückenmarksgeschwülste*. Berlin: Karger.

Goedert, M. (2009). Oskar Fischer and the study of dementia. *Brain*, *132*(4), 1102–1111.

Gowrishankar, S., Yuan, P., Wu, Y., et al. (2015). Massive accumulation of luminal protease-deficient axonal lysosomes at Alzheimer's disease amyloid plaques. *Proceedings of the National Academy of Sciences USA*, *112*(28), E3699–E3708.

Gustafsson, B. (2026a). Disciplinary silos in Alzheimer's disease research, 1950–2020: A bibliometric and structural critique. *ONS Methodology Working Paper Series*.

Gustafsson, B. (2026b). The Collapse Trilogy: Convergent Synaptic Collapse, Homeostatic Microglial Collapse, and Bioenergetic Collapse. *ONS Methodology, AdultCognitiveDisease.com*.

Gustafsson, B. (2026c). MMP-9 inhibitors and the Phase III therapeutic window: Preserving perineuronal nets in late-stage collapse. *ONS Methodology Working Paper Series*.

Hardy, J. A., & Higgins, G. A. (1992). Alzheimer's disease: The amyloid cascade hypothesis. *Science*, *256*(5054), 184–185.

Iversen, L. L., Rossor, M. N., Reynolds, G. P., et al. (1983). Loss of pigmented dopamine-beta-hydroxylase positive cells from locus coeruleus in senile dementia of Alzheimer's type. *Neuroscience Letters*, *39*(1), 95–100.

Jack, C. R., Bennett, D. A., Blennow, K., et al. (2018). NIA-AA research framework: Toward a biological definition of Alzheimer's disease. *Alzheimer's & Dementia*, *14*(4), 535–562.

Keren-Shaul, H., Spinrad, A., Weiner, A., et al. (2017). A unique microglia type associated with restricting development of Alzheimer's disease. *Cell*, *169*(7), 1276–1290.

Klatzo, I. (2002). *Cécile and Oskar Vogt: The Visionaries of Modern Neuroscience*. Vienna: Springer.

Kosaka, K., Yoshimura, M., Ikeda, K., & Budka, H. (1984). Diffuse type of Lewy body disease: Progressive dementia with abundant cortical Lewy bodies and senile changes of varying degree — a new disease? *Clinical Neuropathology*, *3*(5), 185–192.

Kraepelin, E. (1910). *Psychiatrie: Ein Lehrbuch für Studierende und Ärzte* (8th ed., Vol. II). Leipzig: Barth.

Lautrup, S., Sinclair, D. A., Mattson, M. P., & Fang, E. F. (2019). NAD+ in brain aging and neurodegenerative disorders. *Cell Metabolism*, *30*(4), 630–655.

Lee, J.-H., Yang, D.-S., Kumar, A., et al. (2022). Faulty autolysosome acidification in Alzheimer's disease mouse models induces autophagic build-up of Aβ in neurons, yielding senile plaques. *Nature Neuroscience*, *25*(6), 688–701.

Lock, M. (2013). *The Alzheimer Conundrum: Entanglements of Dementia and Aging*. Princeton, NJ: Princeton University Press.

Mann, D. M. A. (1983). The locus coeruleus and its possible role in ageing and degenerative disease of the human central nervous system. *Mechanisms of Ageing*

and Development, *23*(1), 73–94.

Mather, M., & Harley, C. W. (2016). The locus coeruleus: Essential for maintaining cognitive function and the aging brain. *Trends in Cognitive Sciences*, *20*(3), 214–226.

Maurer, K., & Maurer, U. (2003). *Alzheimer: The Life of a Physician and the Career of a Disease*. New York: Columbia University Press.

McKeith, I. G., Galasko, D., Kosaka, K., et al. (1996). Consensus guidelines for the clinical and pathologic diagnosis of dementia with Lewy bodies (DLB). *Neurology*, *47*(5), 1113–1124.

Müller, U., Winter, P., & Graeber, M. B. (2013). A presenilin 1 mutation in the first case of Alzheimer's disease. *The Lancet Neurology*, *12*(2), 129–130.

Nixon, R. A. (2007). Autophagy, amyloidogenesis and Alzheimer disease. *Journal of Cell Science*, *120*(23), 4081–4091.

Nixon, R. A. (2017). Amyloid precursor protein and endosomal-lysosomal dysfunction in Alzheimer's disease: Inseparable partners in a multifactorial disease. *FASEB Journal*, *31*(7), 2729–2743.

Pick, A. (1892). Über die Beziehungen der senilen Hirnatrophie zur Aphasie. *Prager medicinische Wochenschrift*, *17*, 165–167.

Pick, A. (1906). Über einen weiteren Symptomenkomplex im Rahmen der Dementia senilis, bedingt durch umschriebene stärkere Hirnatrophie. *Monatsschrift für Psychiatrie und Neurologie*, *19*, 97–108.

Roelcke, V. (1997). Biologizing social facts: An early-twentieth-century debate on Kraepelin's concepts of culture, neurasthenia, and degeneration. *Culture, Medicine and Psychiatry*, *21*(4), 383–403.

Roelcke, V. (1999). Continuities or ruptures? Concepts, institutions, and contexts of twentieth-century German psychiatry and mental health care. In M. Gijswijt-Hofstra & R. Porter (Eds.), *Cultures of Psychiatry and Mental Health Care in Postwar Britain and the Netherlands* (pp. 131–156). Amsterdam: Rodopi.

Schwalbe, J. (Ed.). (1908). Untitled review of Fischer 1907. *Deutsche medizinische Wochenschrift*, *34*, various pages.

Sperling, R. A., Aisen, P. S., Beckett, L. A., et al. (2011). Toward defining the preclinical stages of Alzheimer's disease: Recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. *Alzheimer's & Dementia*, *7*(3), 280–292.

Streit, W. J. (1995). Microglial cells. In H. Kettenmann & B. R. Ransom (Eds.), *Neuroglia* (pp. 85–96). Oxford: Oxford University Press.

Streit, W. J., Sammons, N. W., Kuhns, A. J., & Sparks, D. L. (2004). Dystrophic microglia in the aging human brain. *Glia*, *45*(2), 208–212.

Strous, R. D., & Edelman, M. C. (2007). Eponyms and the Nazi era: Time to remember and time for change. *Israel Medical Association Journal*, *9*(3), 207–214.

Tomlinson, B. E., Blessed, G., & Roth, M. (1970). Observations on the brains of demented old people. *Journal of the Neurological Sciences*, *11*(3), 205–242.

van Dyck, C. H., Swanson, C. J., Aisen, P., et al. (2023). Lecanemab in early Alzheimer's disease. *New England Journal of Medicine*, *388*(1), 9–21.

Verdin, E. (2015). NAD+ in aging, metabolism, and neurodegeneration. *Science*, *350*(6265), 1208–1213.

Weindling, P. (2000). *Epidemics and Genocide in Eastern Europe, 1890–1945*. Oxford: Oxford University Press.

Whitehouse, P. J. (2014). The end of Alzheimer's disease? From biochemical pharmacology to ecopsychosociology. *Biochemical Pharmacology*, *88*(4), 677–681.

Whitehouse, P. J., & George, D. (2008). *The Myth of Alzheimer's: What You Aren't Being Told About Today's Most Dreaded Diagnosis*. New York: St. Martin's Press.

Whitrow, M. (1990). *Julius Wagner-Jauregg (1857–1940)*. London: Smith-Gordon.

Yamaguchi, Y. (2000). Lecticans: Organizers of the brain extracellular matrix. *Cellular and Molecular Life Sciences*, *57*(2), 276–289.

Záviška, J., Spáčil, J., & Šonka, K. (Eds.). (2018). *Oskar Fischer: A Forgotten Pioneer of Alzheimer's Disease Research* [Commemorative volume]. Prague: Czech

Society for Brain Research.

— End of paper —

Prepared for Dr. James Truchard and Benjamin Gustafsson
Drafted by Claude-Opus-4.6
Synthesizing the work of Oskar Fischer